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ENVIRONMENTAL TOBACCO SMOKE AND PUBLIC HEALTH

This paper describes the existing scientific evidence around the world regarding ETS and the public health. It summarizes the numerous studies regarding ETS and lung cancer, and it describes the extensive research that has been conducted on such health issues as heart disease, respiratory conditions, asthma, and allergies. It assesses the current state of scientific knowledge, and explains the implications of that knowledge for public policy. It does not claim to represent the final word on the issue -- science is never static, and much more research is yet to be performed. It does, however, provide a balanced perspective on what is all too often presented as a one-sided and distorted discussion of ETS and public smoking.

NONSMOKERS' EXPOSURE TO TOBACCO SMOKE

Public debates about ETS are sometimes dominated by anecdotes about individuals who sit for several hours in tiny smoke-filled chambers where a series of machines puff away on an endless series of cigarettes. In this as in all areas, however, public policy must be based on typical conditions in everyday life -- not on exceptional conditions that rarely recur. A first question, therefore, is how much exposure to ETS do typical nonsmokers really experience?

For several years a number of scientists have sought to estimate exposure to ETS by observing and testing everyday working environments. Although much work remains to be done and serious questions exist about some of the methodologies that have been used, even the studies hostile to smoking generally show that nonsmokers typically have only minimal exposure to ETS. Many such studies draw comparisons with active

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smokers which, although questionable as a matter of scientific methodology, still illustrate how very small is the typical exposure. For example, a recent study in Canada showed that a nonsmoker would have to spend 260 hours in an average office to be exposed to the amount of nicotine contained in one cigarette.¹

This result was not in any way atypical: other studies have shown that on average a nonsmoker is exposed to no more than one-quarter of one percent of the smoke inhaled by an active smoker.² A report of the U.S. National Academy of Sciences compared ETS exposure over 8 hours with the exposure of a pack-a-day smoker, and concluded that as a result of ETS exposure a nonsmoker might inhale "approximately 0.005% to 0.26% of the amount of tar deposited in the active smoker's lungs after smoking 20 cigarettes."³

Faced with these facts, smoking opponents have argued that ETS is somehow more toxic than the smoke inhaled directly by smokers. They claim that the smoke from the burning end of a cigarette -- called "sidestream" smoke -- contains higher amounts of some potentially harmful constituents than the "mainstream" smoke inhaled by smokers. To support this assertion, they have relied on laboratory tests using smoking machines and enclosed chambers, where smoke constituents are measured at a distance of only a few centimeters from the end of a burning cigarette.

Such experiments are simply not realistic, however. In normal life, sidestream smoke is not collected in small chambers; instead, it initially exists only in small amounts and then is immediately diluted in the surrounding air. In fact, scientists estimate

1. J. Carson and C. Erickson, "Results From Survey of Environmental Tobacco Smoke in Ottawa, Ontario," Env. Technology Letters 9 (No. 6) 501-08 (June 1988).

2. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects 125-126(1986); A. Arundel, T. Irwin, and T. Sterling, "Non-smoker Lung Cancer Risks from Tobacco Smoke Exposure: An Evaluation of Repace and Lowrey's Phenomenological Model," J. Environmental Science & Health 4:93-118 (1986).

3. Id., Committee on Passive Smoking.

that under such normal conditions, ETS constituents such as nicotine exist in amounts equal to less than 1/100th of those found in mainstream smoke.⁴

As the foregoing discussion suggests, inhaling ETS is far different than smoking a cigarette. Although more needs to be learned about these differences, it is quite clear that the levels of smoke constituents that one encounters in ETS are extraordinarily low compared to active smoking.

ETS AND CANCER

Of all of the health claims made about ETS, none has received greater attention than the possibility of a link with cancer -- both lung cancer and other forms as well. It would be unfair to assert that this research has reached its final culmination: there is a need for ongoing work, which will continue to add to our understanding of ETS. At present, however, there is no persuasive evidence that exposure to ETS significantly increases the risk of cancer. As the American Cancer Society in the U.S. recently recognized with respect to lung cancer, "the currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers * * *"⁵

Lung Cancer. The American Cancer Society reached its conclusion despite the suggestions in some other studies that exposure to ETS can increase a nonsmoker's risk of contracting lung cancer by 35 to 40 percent.⁶ This fact underscores one of the principal difficulties in making sense of existing scientific research. In any area of scientific research there are frequently conflicting studies and inconclusive results -- the recent, dramatic public debate about nuclear "cold" fusion provides a perfect example. It is therefore essential

4. M. Muramatsu, et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch. Occup. Environ. Health 59: 545-50 (1987); M. Muramatsu, et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ. Research 35: 218-27 (1984).

5. American Cancer Society, General Facts on Smoking and Health 3 (1988).

6. United States Department of Health and Human Services, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General (1986); Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects (1986).

that scientists follow fundamental maxims of research, reaching conclusions only after considering all the factors that may contribute to the development of a disease such as lung cancer. As discussed below, when all of these relevant factors are taken into account, the only fair conclusion, as the Cancer Society found, is that the supposed link between ETS and lung cancer remains unproven.

The discussion in the scientific community began in earnest in 1981. That year witnessed the publication of two studies that received widespread attention -- one by Dr. Takeshi Hirayama of the National Cancer Centre Research Institute in Japan,⁷ and another by Dr. Dimitrios Trichopoulos at the University of Athens in Greece.⁸ Both studies purported to find that nonsmoking wives of smokers face an increased risk of lung cancer.

Since the publication of these two studies, however, most subsequent studies have concluded that there is no statistically-significant relationship between ETS and lung cancer. Numerous scientists have concluded that the early Hirayama and Trichopoulos studies were both marred by errors which rendered their conclusions unreliable. Once these errors are corrected, the available evidence fails to prove any significant relationship between ETS and lung cancer.

Virtually all scientists now agree that a study of ETS will be reliable only if it controls carefully for all other important variables that might cause lung cancer. These factors include poor diet and fitness, and occupations that expose workers to indoor and outdoor air pollution. These variables are particularly important because individuals who smoke are not randomly distributed in society. In some countries, families with smokers tend to fall disproportionately in lower income classes, where diet is often not as healthy and there is a greater chance of employment in an occupation with exposure to air pollution.

7. T. Hirayama, "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer," Br. Med. J. 1, 282: 183-85 (1981).

8. D. Trichopoulos, et al., "Lung Cancer and Passive Smoking," Int. J. Cancer 27(1): 1-4 (1981).

Researchers have found that the original Hirayama and Trichopolous studies were biased by a failure to account properly for these other variables. For example, a recent Hong Kong study showed that the wives of smokers were typically less wealthy and had less "healthy" lifestyles than the wives of nonsmokers. The wives of smokers had diets consisting of more processed foods and fewer fresh fruits and vegetables. As a result, according to this research, a failure to account for these factors biased Hirayama's research results.⁹

In addition, scientists have found that lung cancer -- like all cancers -- tends most often to afflict those of an advanced age. Thus, when comparing two population groups, one must compare groups with closely similar age distributions. In 1988, two reports for a conference in the United Kingdom found that the Hirayama study had failed to divide the study population into appropriate age groups. The reports found that when such age bias was removed, the purported risk to nonsmoking wives disappeared completely.¹⁰

Further, exposure to air pollution may be a significant factor in biasing research on the causes of lung cancer. For example, one recent study of Chinese women found a significant correlation between the risk of lung cancer and the extent to which the women had been exposed on an extended basis to cooking oil vapors.¹¹ Because the 1981 Hirayama and Trichopolous studies failed to account for exposure to air pollution, they ignored an important variable that may have substantially distorted the research findings.

Another basic flaw in the Hirayama study was the absence of reliable information on smoking status. The Hirayama study determined the smoking habits of women and their husbands by use of a questionnaire. The subjects were polled only once,

9. L.C. Koo, "Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked," Nutr. Cancer 11: 155-72 (1988).

10. W. Ahlborn and K. Uberla, "Passive Smoking and Lung Cancer: Reanalyses of Hirayama's Data," in Indoor and Ambient Air Quality (R. Perry and P.W. Kirk, eds.) 169-78 (London: Selper Ltd., 1988); S.J. Kilpatrick and J. Viren, "Age as a Modifying Factor in the Association Between Lung Cancer in Non-smoking Women and Their Husbands' Smoking Status," in Indoor and Ambient Air Quality, 195-202.

11. Gao, et al., "Lung Cancer Among Chinese Women," Int. J. Cancer 40: 604-09 (1987).

with no effort to update the original data. Thus, if someone stopped or started smoking even a week after completing the questionnaire, this was never discovered or considered.

Hirayama also made no effort to verify the accuracy of the questionnaire's classification of nonsmokers. A British researcher, Peter Lee, has published a series of papers studying potential misclassification problems in questionnaires that ask respondents to state whether they smoke.¹² He has found that up to ten percent of subjects who claimed never to have smoked made inconsistent statements on other occasions, and that the percentage is even higher in questionnaires that ask whether individuals have stopped smoking.¹³ Lee found that even if only five percent of the reported nonsmokers had answered a questionnaire incorrectly, this would lead researchers falsely to conclude that ETS increased the risk of developing lung cancer by 44 percent.¹⁴ In fact, for women married to husbands who smoked one to 14 cigarettes per day, the Hirayama study concluded that ETS increased the risk of lung cancer by 42 percent; for women married to husbands who smoked 15-19 cigarettes per day, Hirayama estimated that the risk increased by 58 percent. In other words, inaccurate responses to Hirayama's questionnaires could account for virtually the entire increase in the reported risk of developing lung cancer.

Research into the health effects of ETS continues, and some of the more recent studies have made important advances in improving upon the methodologies utilized in the Hirayama and Trichopolous studies. These include two recent studies that, for the first time, have incorporated microscopic work to identify the types of cancers involved. Dr. Linda C. Koo at the University of Hong Kong has headed a series of research efforts that have combined the work of a pathologist to identify cancers with strict controls to sep-

12. The conclusions from these papers have recently been published in a monograph. P. Lee, Misclassification of Smoking habits and Passive Smoking: A Review of the Evidence (Int. Archives of Occ. and Environ. Health Supp. 1988).

13. *Id.* at 62.

14. *Id.*

arate the effects of other variables such as age and diet.¹⁵ The research by Dr. Koo and her colleagues led to several conclusions. Most important, it determined that when problems of misdiagnosis and independent variables were removed, ETS exposure in the home and at work had no statistically significant impact on the incidences of lung cancer.¹⁶

Researchers with the American Cancer Society in New York have conducted similarly thorough cancer research. A team of researchers headed by Dr. Lawrence Garfinkel worked with a pathologist who reviewed for each patient a series of medical slides of actual cancer cells. This review showed that overall there was no statistically significant increase in the risk of lung cancer for nonsmoking women married to smokers. Indeed, ETS caused even a marginally significant risk increase for only one subgroup -- women married to men who smoke more than 20 cigarettes per day.¹⁷

These recent studies cast substantial doubt on the validity of the 1981 research that sparked the ongoing debate on the effects of ETS. In a 1987 article, a German scientist reviewed all of the research conducted since 1981 and concluded that "the major trend of the evidence" does not support the argument that ETS causes lung cancer.¹⁸ Indeed, the article noted that "all studies with positive associations [between ETS and lung cancer] can just as well be explained by chance, bias, confounding or misclassification."¹⁹ Joining with the conclusion of others, this further indicates, as the American Cancer Soci-

15. L.C. Koo, et al., "Is Passive Smoking an Added Risk for Lung Cancer in Chinese Women?" J. Experimental and Clinical Cancer Research 3(3): 277-83 (1984); L.C. Koo, et al., "Measurements of Passive Smoking and Estimates of Lung Cancer Risk Among Non-Smoking Chinese Females," Int. J. Cancer 39: 162-69 (1987); L.C. Koo, et al., "Life-History Correlates of Environmental Tobacco Smoke: A Study on Nonsmoking Hong Kong Chinese Wives with Smoking Versus Nonsmoking Husbands," Soc. Sci. Med. 26(7): 751-60 (1988); L.C. Koo, "Dietary Habits and Lung Cancer Risk Among Chinese Females in Hong Kong Who Never Smoked," Nutr. Cancer 11: 155-72 (1988).

16. Id.

17. L. Garfinkel, et al., "Involuntary Smoking and Lung Cancer," J. Nat. Cancer Inst. 75(3): 463-69 (1985).

18. K. Uberla, "Lung Cancer From Passive Smoking: Hypothesis or Convincing Evidence?" Int. Archives of Occ. and Environ. Health 59: 421-37 (1987).

19. Id.

ety concluded, that "the currently available evidence is not sufficient to conclude that passive or involuntary smoking causes lung cancer in nonsmokers * * * "20

Other Cancers. In comparison to lung cancer, scientists have devoted less attention to the role of ETS in other types of cancer. This is not surprising since -- in comparison to the lungs -- the other parts of the human body are not as directly exposed to tobacco smoke. Thus, it seems even less likely that ETS could play a strong role in causing cancer in other parts of the body.

Dr. Hirayama has devoted the most attention to other types of cancers. His research showed no relationship between ETS and cervical cancer, but purportedly higher risks for cancers in the nose and brain.²¹ Other scientists, however, have found the suggestion of such increased risks to be counter-intuitive at best. Scientists have not found that active smoking increases the risk of brain or nose tumors. Therefore, it seems highly implausible that non-smokers exposed to the low levels of constituents found in ETS should confront higher risks of such diseases.

A more logical explanation for Hirayama's conclusion lies once again with the flaws in his underlying research. As with the conclusions on lung cancer, Hirayama's data are distorted by the failure to account for other factors and the strong prospect of misreporting on questionnaire responses. There is thus good reason to reject Hirayama's conclusions, as did the U.S. National Research Council in 1986. The Council reviewed all of the available studies and concluded that "there is no consistent evidence at this time of any increased risk from ETS exposure for cancers other than lung cancer."²²

OTHER DISEASES

20. American Cancer Society, General Facts on Smoking and Health 3 (1988).

21. T. Hirayama, "Non-Smoking Wives of Heavy Smokers Have a Higher Risk of Lung Cancer," Br. Med. J. I, 282: 183-85 (1981).

22. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects (1986).

Scientists have also focused their attention on the possibility of a relationship between ETS and other types of diseases. As with cancer, however, there is no conclusive evidence that ETS actually plays any significant role in causing other diseases.

Heart Disease. Numerous scientists have examined the possibility of a relationship between ETS and heart disease. Their research does not support a causal link between ETS exposure and heart disease -- a fact recognized even by the U.S. National Academy of Sciences. According to the National Academy's 1986 report which reviewed all available research, there is no evidence among healthy individuals of any "statistically significant effects due to ETS exposure."²³ Nor is there any "evidence of serious harm in people with heart disease."²⁴

Several scientific studies have evaluated the possible effects of ETS on healthy children and adults. The 1986 National Academy of Sciences report reviewed five such studies that evaluated individuals with and without exposure to ETS. According to the report, the studies showed that "[t]here were no significant changes * * * in heart rate or blood pressure in school-aged children or in adult men and women."²⁵ The National Academy also concluded that "[d]uring exercise there was no difference in the cardiovascular changes for men and women between conditions of exposure to ETS and control conditions."²⁶

Scientists also have evaluated whether exposure to ETS causes increases in carbon monoxide levels in the blood. High carbon monoxide levels -- if found -- could increase the risk of heart disease. Four scientific studies have measured the effects of ETS in a variety of locations, including aircraft, restaurants, offices, and even the closed envi-

23. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects 265 (1986) [hereinafter National Academy Report].

24. Id. at 266.

25. Id. at 259.

26. Id. at 265.

ronment of a submarine.²⁷ These studies have failed to detect any increase in carbon monoxide levels in non-smokers. Similarly, the National Academy of Sciences in 1986 reviewed several studies that had analyzed the effects of ETS in experimental situations.²⁸ These showed an average increase in carbon monoxide of less than one-half of one percent. This is less than one-twentieth of the increase sometimes observed in active smokers.

In light of these studies, two German researchers concluded in 1984 that "there is little evidence" to suggest that substances found in ETS "may adversely affect the cardiovascular system."²⁹ This echoed the conclusions of a Geneva Symposium on ETS one year earlier, which noted that "carbon monoxide from environmental tobacco smoke is not important from a health point of view."³⁰

Finally, scientists have conducted epidemiological studies to assess whether ETS increases the risk of heart disease. These provide conflicting results. Three studies since 1984 have found that there is no significant association between ETS and heart disease.³¹ Nonetheless, some antismokers have argued that two other studies support a con-

27. M.J. Jarvis, et al., "Absorption of Nicotine and Carbon Monoxide from Passive Smoking Under Natural Conditions of Exposure," Thorax 38: 829-33 (1983); D. Folliart, et al., "Passive Absorption of Nicotine in Airline Flight Attendants," N. Engl. J. Med. 308: 1105 (1982); A. Seppanen, et al., "Carboxyhemoglobin Saturation in Relation to Smoking and Various Occupational Conditions," Ann. Clin. Res. 9: 201-68 (1977); N.F. Lightfoot, "Chronic Carbon Monoxide Exposure," Proc. R. Soc. Med. 65: 798-99 (1972).

28. National Academy Report, at 258, evaluating T.E. Dahms, et al., "Passive Smoking: Effects on Bronchial Asthma," Chest 80: 530-34 (1981); R. Huch et al., "Risks the Passive Smoker Runs," Lancet 2: 1376 (1980); C. Hugod, et al., "Exposure of Passive Smokers to Tobacco Smoke Constituents," Int. Arch. Occup. Environ. Health 41: 145-49 (1978); P.E. Pimm, et al., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Arch. Environ. Health 33: 201-13 (1978); E. Polak, "Le papier a cigarette. Son role dans la pollution des lieux habites. Tabagisme passif: Notion nouvelle precise," Bru. Med. 57:335-40 (1977); A. Seppanen, et al., "Carboxyhemoglobin Saturation in Relation to Smoking and Various Occupational Conditions," Ann. Clin. Res. 9: 201-68 (1977).

29. H. Schievelbein and F. Richter, "The Influence of Passive Smoking on the Cardiovascular System," Prev. Med. 13(6): 626-44 (1984).

30. R. Rylander, "Workshop Perspectives," ETS-Environmental Tobacco Smoke: Report From a Workshop on Effects and Exposure Level (R. Rylander, et al, eds.), in Eur. J. Respir. Dis. (Suppl. 133(65): 143-45 (1984).

31. P. Lee, et al., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," Brit. J. Cancer 54: 97-105 (1986); C. Garland, et al., "Effects of Passive Smoking on Ischemic Heart Disease Mortality of Nonsmokers," Am. J. Epidemiol. 121(5): 645-50 (1985); C. Gillis, et al., "The Effect of Environmental Tobacco Smoke in Two Urban Communities in the West of Scotland," in ETS-Environmental To-

trary view. In the first study,³² however, all of the data showed either that ETS had no significant effect or only slight effects that were of no more than borderline (and therefore questionable) significance. The second study relied on a 1963 census in a Maryland county and subsequent review of death certificates.³³ This study reported a small increase in the risk of heart disease among nonsmokers living with smokers, but qualified this finding because of the researchers' inability to account for changes in behavior over time and other important factors. As the authors themselves noted, "other factors such as diet and exercise might differ in families with and without smokers; we cannot ignore the possibility that such differences could influence our findings."³⁴

In sum, the epidemiological evidence supporting a causal link between ETS exposure and heart disease remains very weak. The studies that might be used to show such a link must be either qualified severely or rejected outright because of conflicting results or the failure to isolate all of the important variables. Moreover, these reports must be compared to the three other studies that show, in the words of the National Academy of Sciences, "no evidence of statistically significant effects due to ETS exposure" * * *³⁵

Respiratory Symptoms. Although other health issues have not received the same attention as cancer and heart disease, five scientific studies have addressed the relationship in adults between ETS and respiratory problems. On balance, these studies fail to show a causal relationship between exposure to ETS and respiratory ailments in otherwise-healthy adults.

Passive Smoke: Report From a Workshop on Effects and Exposure Level (R. Rylander, et al, eds.), in Eur. J. Respir. Dis. (Suppl. 133(65): 121-26 (1984).

32. K. Svendsen, et al., "Effects of Passive Smoking in the Multiple Risk Factor Intervention Trial," Am. J. Epidemiology 126(5): 783-95 (1987).

33. K.J. Helsing, "Heart Disease Mortality in Nonsmokers Living With Smokers," Am. J. Epidemiology 127(5): 915-22 (1988).

34. Id.

35. National Academy Report, supra, at 265.

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Three major studies have found that there is no "statistically significant reduction of pulmonary function in passive smokers."³⁶ A 1988 update of one of these studies further confirmed this view, concluding that "there is no evidence that average everyday passive smoke exposure in the office or at home leads to an essential reduction of lung function in healthy adults."³⁷

Antismoking groups frequently respond to this research by arguing that a 1980 study³⁸ supports a connection between ETS and respiratory problems in the workplace. A closer review of the evidence, however, shows that this is not the case. This study contained numerous flaws, including nonstandardized questionnaires and unsupervised reporting. These and other problems led even the U.S. Surgeon General to criticize the report, stating that its classification procedures were "crude and did not account for people who changed jobs. It is unclear how the ex-smokers in the population were handled in the analysis."³⁹

The other principal study cited by antismokers⁴⁰ contains comparable flaws -- including data results that seem to be simply implausible. For example, based on this survey, it appears that men exposed to light amounts of ETS actually have better respiratory symptoms than men who are exposed to no ETS at all.

Even the U.S. Government has twice concluded that there is no convincing evidence that ETS causes lung problems in adults. In 1983 a Government workshop found

36. M. Kentner, et al., "The Influence of Passive Smoking on Pulmonary Function - A Study of 1351 Office Workers," Prev. Med. 13: 656-69 (1984), referring to R.S.F. Schilling, et al., "Lung Function, Respiratory Disease, and Smoking in Families," 106: 274:83 (1977), and G.W. Comstock, et al., "Respiratory Effects of Household Exposure to Tobacco Smoke and Gas Cooking," Am. Rev. Respir. Dis. 142(2): 143-48 (1981).

37. M. Kentner and D. Wetle, "Passive Tobacco Smoke Inhalation and Lung Function in Adults," Indoor and Ambient Air Quality (R. Perry and P.W. Kirk eds.) 232-41 (1988).

38. J.R. White and H.F. Froeb, "Small-Airways Dysfunction in Non smokers Chronically Exposed to Tobacco Smoke," N. Engl. J. Med. 302: 702-23 (1980).

39. United States Department of Health and Human Services, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General 60 (1986).

40. F. Kauffmann, et al., "Adult Passive Smoking in the Home Environment: A Risk Factor for Chronic Airflow Limitation," Am. J. Epidemiol. 117: 269-80 (1983).

that the available research on "the effect of passive smoking on the respiratory system suggests that the effect varies from negligible to quite small."⁴¹ Three years later even the U.S. Surgeon General concurred, stating that "[t]he small magnitude of effect implies that a previously healthy individual would not develop chronic lung disease solely on the basis of involuntary tobacco smoke exposure in adult life."⁴²

Asthma. Only a few studies have assessed the possibility of a relationship between ETS exposure and asthma. Although anecdotal accounts abound -- both supporting and rejecting such a relationship -- at present there exists no persuasive scientific evidence in support of any conclusion on the issue.

The four existing studies on asthma restricted themselves to only a small number of subjects -- varying from only 14 to 23 patients.⁴³ These studies exposed asthmatic patients to ETS in a closed chamber, typically for an extended period of time. The most recent (and sophisticated) of these studies found that despite a "severe simulation of passive smoking, beyond what normally occurs in the majority of social or occupational environments," exposure to ETS caused "no acute respiratory risk" to asthmatic patients.⁴⁴

The other three studies varied, with two finding no relationship between ETS and asthma and the third⁴⁵ -- with 16 patients -- finding that ETS increased asthmatic conditions. As the Surgeon General stated, however, in the latter study the test "subjects were put in a small chamber, were "not blinded as to the exposure and were selected be-

41. United States Department of Health and Human Services, Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiological Studies, May 1-3, 1983 (1983).

42. United States Department of Health and Human Services, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General 62 (1986).

43. H.P. Weidemann, et. al, "Acute Effects of Passive Smoking on Lung Function and Airway Reactivity in Asthmatic Subjects," Chest 89(2): 180-85 (Feb. 1986); T.E. Dahms, et. al, "Passive Smoking: Effects on Bronchial Asthma," Chest 80(5): 530-34 (1981); R.J. Shepard, et. al, "Passive Exposure of Asthmatic Subjects to Cigarette Smoke," Env. Research 20(2): 392-402 (1979); ; P.E. Pimm et. al, "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Archives of Env. Health 33(4): 201-13 (1978).

44. Weidemann, *supra*.

45. Dahms, *supra*.

cause of complaints about smoke sensitivity.⁴⁶ In light of these biases, it is hardly surprising that this research showed a much stronger causal relationship than the others.

In the final analysis, it is simply not possible to reach any definitive conclusions about ETS and asthma. The number of examined patients has been too small, and the experimental conditions too unrealistic. While the weight of existing evidence indicates that ETS poses no additional significant risk for individuals with asthma, the final word must await additional research.

Allergies. Finally, vocal complaints about the sight and smell of tobacco smoke have prompted some to speculate that some individuals might actually be allergic to ETS. Nonetheless, despite these complaints, scientists have not been able to confirm that any such allergies exist. For example, a 1980 study determined that "direct evidence that tobacco smoke is immunogenic [capable of causing a specific response] in man is yet to be determined."⁴⁷ Another more recent study, completed in 1986, reached this same conclusion.⁴⁸

The primary research in support of allergic reactions to ETS was based on the use of tobacco leaves, not tobacco smoke.⁴⁹ This research exposed test subjects to tobacco leaf extract and tested for allergic skin responses. The flaw in this approach, however, was documented by an English immunologist who pointed out the "great difficulties" involved in predicting allergies to tobacco smoke on the basis of reactions to tobacco

46. Surgeon General's Report, *supra*, at 65.

47. S. Lehrer, et. al, "Immunogenicity of Tobacco Smoke Components in Rabbits and Mice," *Int. Arch. Allergy Appl. Respir.* 62: 16-22 (1980).

48. S. Lehrer, et. al, "Tobacco Smoke Sensitivity: A Result of Allergy?," *Ann. Allergy* 56: 1-10 (1986).

49. G. Bylin, "Tobaksallergi - finns den?," *Läkartidningen* 77(16): 1530-32 (1980); K. Tibbling, "Elicitation of Bronchial Asthma with Tobacco Extract," *Abstracts of the 4th World Conference on Smoking and Health* (June 18-21, 1979); I. Ståhle and L. Tibbling, "Tobaksallergi hos Patienter Med Asthma Bronchiale," *Läkartidningen* 75(17): 1711-13 (1978).

leaves.⁵⁰ Recent research confirms this point, showing that there is no association between tobacco leaf sensitivity and respiratory difficulties in allergic asthmatics.⁵¹

As indicated by this review, despite the broad assertions of antismoking groups, there simply is no firm scientific basis for concluding that ETS causes health problems for nonsmokers. Scientific research should and will continue. Increasingly, however, this additional research underscores the absence of health problems resulting from ETS -- and not the other way around. Thus, it is simply unfair to claim -- as antismoking groups increasingly do -- that other peoples' tobacco smoke presents a danger for nonsmokers.

50. G. Taylor, "Tobacco Smoke Allergy -- Does It Exist?," Scand. J. Respir. Dis. Suppl. 91: 50-55 (1974).

51. R.P. Stankus and S.B. Lehrer, "Inhalation Challenge Studies on Tobacco Smoke Sensitive Asthmatics," in Indoor and Ambient Air Quality (R. Perry and P.W. Kirk eds.) 303-04 (1988).